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Neurosciences

Buying Time for Damaged Tissue

Researchers hope a new drug called Nimodipine will reduce the severity of strokes.

Nimodipine, a calcium channel blocker, is now being clinically tested at the Medical College of Georgia and at other sites in the United States.

When cells are severely damaged, calcium filters in through disrupted membranes. The cells are then destroyed by enzymatic reactions set up by the presence of calcium.

If calcium can be kept out of the cells, some researchers believe it may buy time to salvage the damaged tissue. "These researchers believe that if the calcium had not gotten into these cells, some of the cells may have recovered," said Dr. Fenwick Nichols, assistant clinical professor of neurology and principal investigator in the Nimodipine study at the Medical College of Georgia.

Nimodipine has been used in trials in Europe, and results there have been promising. One trial used the drug to treat people with sub-arachnoid hemorrhage.

Such a hemorrhage is bleeding between the arachnoid covering and the brain. It is usually caused by an aneurysm or a weak spot on an artery.

A sub-arachnoid hemorrhage initially causes trouble by bleeding but as a complication the patient can have what is called vasospasm, in which the blood vessel contracts, restricts blood flow and often causes a stroke. This is called an ischemic event.

"They gave Nimodipine to a number of people who had this type of hemorrhage and found they had lower incidence of the spasm and fewer ischemic events, complications of the spasm. This suggests this was a good drug for the treatment of patients who had sub-arachnoid hemorrhage," Dr. Nichols said.

The second European study was done on patients with ischemic strokes. Results showed a 21 percent mortality rate in the group given a placebo and only a 7 percent mortality rate in those given Nimodipine, said Dr. Nichols.

The European studies seemed to conclude that patients who survived with Nimodipine had a better functional outcome. "This means they returned more nearly towards normal," Dr. Nichols said. "I'm not saying they got completely normal, but they did better, they appeared to do better, than the people who did not receive the Nimodipine."

The promising results of these studies prompted the Bayer drug company to seek approval of Nimodipine in the U.S. To this end, the two-year, multi-center study, which includes MCG, began.

At the end of the two years, the drug company will analyze the results and compare the drug to the placebo. They will look for two things: whether the drug improved survival rates or functional outcomes and major side effects. The results of the analysis will be presented to the Federal Drug Administration and a ruling made on sale of Nimodipine in the U.S.

Patients in the study receive one of four doses: a placebo or a low, medium or high dose of Nimodipine. Not all stroke victims are eligible for the study, however, Dr. Nichols said.

"To be a possible candidate to receive the drug, the patient must have had what is called an ischemic stroke, one that means you didn't get enough blood to the brain, not a hemorrhage. The patient has to be functional but display a certain amount of weakness," he said. Potential patients can't have serious illnesses or be on any other calcium channel blockers.

Dr. Fenwick Nichols says "ah" in clinic with patient Susan Dickerson.

The drug must be started within 48 hours or at the onset of the stroke. Experimental data shows that the drug is most likely to have a good effect if administered within six hours of the stroke.

The Medical College of Georgia is one of between 40 and 50 centers, mostly teaching hospitals, in the country to

Nimodipine is a drug being clinically tested for use in improving the recovery of stroke patients.

test the drug. About 1,200 patients will be tested in all of the centers.

So far, Dr. Nichols and his group have entered nine people in the MCG study.

"There have been a number of people who could have been entered who just got to the hospital too late," he said.

"They were willing, they had the right kind of stroke but they just didn't get to the hospital in time. That's one of the biggest problems we have, getting folks to come in early. Many times they'll stay at home thinking 'my arm's going to get stronger,' 'my talking's going to get better,' rather than coming directly to the hospital."

Dr. Nichols is quick to point out that Nimodipine does not cure strokes. It only makes them less severe.

But "it's very encouraging when you have something like this," he said.

"For the first time, we have available a medication that appears to be able to decrease the mortality and lessen the severity of a new stroke. It is encouraging that this drug might allow us to immediately intervene in a new stroke and improve the quality of life for the stroke victim." ■

—John Donnelly

Recouping Losses

The rehabilitation of a stroke patient is a multifaceted endeavor.

The patient needs not only medical care but also therapy to restore lost motor skills and cognitive abilities. For the physical therapist, occupational therapist or speech pathologist, evaluation is the first concern. Most of a patient's improvement will occur within six months to a year of the stroke, so therapists must work quickly.

"The first thing we do when we get a patient referral is an evaluation," said Claudia Morin, chief of occupational therapy at the Medical College of Georgia Hospital and Clinics. "This includes gathering data on their occupational performance, what the patient's abilities and limitations are in the areas of self-care, work and leisure, and their component functions, their cognitive ability, sensory motor status and psychosocial skills. The evaluation is geared to finding out what the patient's basic problems are."

While the occupational therapist's evaluation determines the range of activities achievable by the stroke patient, the physical therapist determines the patient's functional performance, overall strength, range of motion and neurological status.

"This includes muscle tone, coordination, abnormal reflexes and the possible neglect of their body. Body neglect is the term applied to a patient who has no awareness of, for example, an arm. They just don't know it's there," said Jeanne Robinson, assistant chief of physical therapy at the Medical College of Georgia Hospital and Clinics.

Verbal and written expression and language comprehension are the chief concerns of Jeannette Young, MCG's speech pathologist, when screening a stroke patient.

"The first thing I try to do is see if the patient has good comprehension or fair comprehension or no comprehension," she said.

Ms. Young realizes the patient may not be able to move much.

"The first thing I might do is ask the patient to close his eyes or look up at the ceiling if he hears me and understands me. If the patient has good comprehension, then the prognosis for recovery of language is better."

People whose strokes have affected the left side of the brain controlling speech almost always have some degree of language comprehension difficulty, she said.

Ms. Young next tests verbal and written expression. The testing includes determining the patient's ability to count and name objects, phrase and put two or three sentences together. "The awful thing about a patient who doesn't have verbal expression is that most often their written expression is also gone," she said.

The health care workers agree that once an evaluation is complete, the next step is getting the patient out of bed as quickly as possible.

"Bed rest is bad," said Ms. Robinson. "As soon as we can, we get stroke patients up and moving against gravity." Getting the patient mobilized helps give him the feeling of normal movement, helps him rediscover muscle coordination and regain a sense of balance. Such movement also helps facilitate the return of normal muscle tone.

"We like patients to get up and move as soon as possible. Lying in bed creates a wonderful environment for sensory deprivation," says Ms. Morin. "If you can get patients upright as soon as possible when they're medically stable, that's almost a 100 percent improvement."

Early interaction with a stroke patient is also recognized as important in speech pathology. "Everybody will agree that with speech intervention, the patient will speak earlier," Ms. Young said. New data from a large Veterans Administration study shows that both the quantity and the quality of recovery are better with early speech intervention.

To rehabilitate a stroke patient, Ms. Robinson uses steps similar to the developmental stages in a baby. "Before a baby is able to walk unassisted, it has used a variety of supports such as furniture or someone's hand, and before getting on its feet, the baby has to develop control of its head and trunk position against gravity. The same is true with a stroke patient."

This involves taking the patient's initial evaluation and working with the patient to improve from this level. "An example would be to have a patient begin walking using the parallel bars and advance to a walker and then a quad-cane (one with four feet instead of one) and then a regular cane," Ms. Robinson said. She adds that each patient's therapy is different. "Each patient is an individual. We don't have a pre-set treatment plan for all the patients."

Once the treatment program is established, the occupational therapist focuses on the basics.

"We look at feeding first, because that is a very important area. Some of the patients are very unaware of their environment and when a food tray is placed in front of them, they don't know what to do with it. One of our prime goals, then, is to foster independence in feeding — chewing and swallowing," Ms. Morin said.

As treatment progresses, patient and therapist work on positioning and manipulating eating utensils during meal time "so that it is an appropriate activity and we are not, in addition to the stress of their disability, imposing a totally unfamiliar kind of situation on the patient," Ms. Morin said.

Another goal of rehabilitation is always to challenge the patient to do more.

"When the patient achieves something, we get them doing something new," Ms. Robinson said. "While they are learning the new activity, we are also reinforcing what has come before, what they are doing well."

The patient's own motivation, not the therapist's, ultimately determines his progress. "A lot of what a patient accomplishes occurs outside of therapy," Ms. Robinson said. "Motivating people is a big part of the job."

Ms. Morin agreed. "We try to increase that motivation within the patient to do things for themselves. Occupational therapy is more an opportunity provider — providing challenges and opportunities for the patient, guiding and grading activities so that the patient is constantly stimulated but not frustrated."

Part of the therapist's role,

Patient Rosa Wright works with Jeanne Robinson (above) measuring range of motion and with Claudia Morin (below) completing a manual task.

Therapists may make the difference when stroke patients are trying to recover lost motor skills and cognitive abilities.

she added, is gauging what a patient can and can't do.

The therapist and the patient are not the only ones who help with motivation.

"One of the most important ingredients in a patient's recovery is the involvement of the family. If they are positive, supportive people, then the patient is going to progress at a faster rate," Ms. Morin said. "This means that we have to educate and train the families in ways to care for the stroke patient and continue the treatment plan at home."

To this end, a big part of the treatment plan for the occupational therapist is evaluating the patient's home environment. This includes safety precautions including hand rails for getting in and out of the bathtub and energy conservation techniques such as rearranging the kitchen to be more accessible.

The speech therapist also must educate the family. "We tell the family the patient hasn't lost his mind, he's lost his language ability. And we can begin work toward his recovery of that function."

"Our long-range goal is to have the stroke patient moving independently in his or her household environment," Ms. Robinson said. "To that end, we work with them until they reach their maximum level of function or until they are discharged from the hospital to a rehabilitation center."

While the family plays a very important part in what happens to the stroke patient after discharge, Ms. Morin feels a holistic approach to outpatient care involving physical therapy and speech therapy personnel is necessary. "That's what we aim to achieve. The essence is that the more people involved in the rehabilitation team, understanding the goals that are laid out for the patient, the better the treatment will progress."

Ms. Young agrees that coordination between the disciplines in formulating treatment plans for outpatients is very important. "This way, when a patient returns for outpatient therapy, we — PTs, OTs and myself — can coordinate our schedules so they get everything they need," she said.

Therapy improves the recovery of many stroke patients, Ms. Robinson said. "We are trying to improve the patient's function and their quality of life. I think we have a good rate of getting the patient to be productive." ■

— John Donnelly

Understanding A Deadly Accomplice

Stroke is usually considered a disease of aging. But children too can fall prey.

Studies show a 5 to 17 percent incidence of stroke in children with sickle cell anemia. It is a major cause of disease and death in both children and adults with the disease. The risk appears greatest in children from age 5 to 15 and the incidence of recurrent stroke after the first episode is high.

The first report linking stroke and sickle cell anemia appeared in 1923 and was written by Dr. Virgil Sydenstricker during his 35 years as chairman of the Department of Medicine at the Medical College of Georgia.

Sickle cell disease is caused by a genetic abnormality in the body's hemoglobin production and appears in such symptoms of anemia as weakness, fatigue and pain, especially in the bones and joints. It is a chronic, not terminal, illness and is almost always found in blacks.

The clinical manifestations range from relatively mild bouts of pain to severe infections and other devastating medical complications.

Why children with sickle cell are susceptible to stroke and how to detect those at risk for stroke are questions that form the basis for a study by Dr. Robert Adams, assistant professor of neurology, and his co-investigators.

Why are these children having strokes, what kinds of strokes are they having, how can they be prevented and treated? The researchers faced these questions as well as the problem of developing a safe way to determine which children with sickle cell anemia are at greatest risk for stroke. They decided to focus on that problem first.

Dr. Robert Adams measures blood flow inside Lakesha Nelson's head.

"Until now, there was no feasible way to determine which children with sickle cell disease were at risk for stroke," Dr. Adams said. "The only thing to do was wait until the child had a stroke and then, using angiography, try to determine the risk for the next one. The problem with this approach is that the first stroke may be very severe and disabling."

Angiography involves injecting a dye through a catheter into the blood stream. An X-ray determines if any abnormalities are present in the area's blood vessels. The procedure is the best way to obtain the desired information, but is expensive, invasive and has a small risk factor, Dr. Adams said.

Angiography is used annually in six to eight young sickle cell anemia patients at the Medical College of Georgia who have had symptoms of cerebrovascular problems (relating to the brain's blood supply) but "is not an acceptable research or screening tool," Dr. Adams said.

Although the cause of stroke in sickle cell children is not known, studies have shown these patients have a narrowing, called cerebrovascular stenosis, of two or more major blood vessels to the brain.

"In 75 percent of sickle cell anemia patients who have had a stroke, the stroke is an infarction stroke where the blood vessels to the brain don't transport enough blood to that organ," said Dr. Fenwick Nichols, assistant clinical professor of neurology and a co-investigator on the study. "The rest of the cases are hemorrhages, where the vessels burst and release blood into the brain. As the children grow

Stroke can be a deadly accompaniment to sickle cell anemia. Researchers are trying to understand why.

older, they become more prone to the hemorrhage type of stroke."

These blood vessels are located at the base of the brain and are relatively inaccessible to non-invasive technologies such as ultrasound because the thickness of the skull prevents the penetration by sound waves.

Within the past five to seven years, however, a type of ultrasound called Transcranial Doppler (TCD) has been used in adults to measure blood flow velocity in the major arteries inside the head. The TCD method uses sound waves to record the shift in the Doppler frequency caused by red blood cells moving through the blood vessels.

"The TCD can pick up partial or complete blockages in these blood vessels," Dr. Adams said. "It uses computer-assisted ultrasound to bounce sound off the red blood cells and determine how fast the blood is flowing."

Dr. Rune Aaslid, the developer of TCD and director of cardiovascular research at the Institute of Applied Physiology and Medicine in Seattle, Washington, is a consultant on the project. "He is the man who first applied this technology, in the early 1980s, to patients who had spasms and a reduction in the size of blood vessels following brain hemorrhages," Dr. Adams said.

When there is an obstruction, the blood moves at an increased velocity. It's like putting your finger over the end of a garden hose — the pressure increases. The volume is the same, but the water flows faster out of a smaller opening.

In a preliminary TCD study using six children, Dr. Adams reported that the technique is at least as feasible in children as in adults despite the fact that the vessels are smaller.

The results of the investigators' early TCD testing have been confirmed by angiography and by magnetic resonance imaging (MRI), according to Dr. Nichols.

Another study previously done by Dr. Adams and Dr. Taher El Gammal, professor of radiology, determined that MRI can detect a narrowing of blood vessels to the brain in sickle cell anemia stroke patients.

An MRI can provide images of the soft tissues inside the skull because it doesn't use radiation, unlike X-rays. It also picks up the outline of blood vessels due to the flow of the blood through them. Although initial results are promising, MRI does not yet provide the same information as angiography.

The MRI, however, has the potential of giving three-dimensional views of blood vessels using advanced computer processing. Although it carries no risk, the cost of an MRI study keeps it from being a cost-effective screening tool. For the study, the screening function will be performed by the TCD.

The researchers will continue to work with Dr. El Gammal in the current study to develop MRI protocols that will more reliably detect areas of blockage in the blood vessels. "This is difficult to do because the 'slices' or images the MRI takes have to be very close together to reveal any blockage," Dr. Adams said.

This project, in fact, began as a pilot study set up by Dr. Adams, Dr. Virgil McKie, assistant professor of pediatrics and cell and molecular biology; and Dr. Alex Felice, assistant professor of cell and molecular biology, when they recognized the importance of cerebrovascular complications in their patients with sickle cell anemia.

The pilot study, supported by the MCG Research Institute and the Comprehensive Sickle Cell Center, set out to determine if MRI technology could be used to evaluate sickle cell anemia stroke patients.

The Comprehensive Sickle Cell Center and its director, Dr. Titus H.J. Huisman, Regents professor, chairman of cell and molecular biology and professor of medicine, supports both research and clinical activities in sickle cell disease. "We hope, working with Dr. Abdulla Kutlar, assistant professor of cell and molecular biology, to bring this expertise to the problem of stroke in these patients through hematological analysis," Dr. Adams said. "Perhaps a clue can be found from the blood as to why some of these patients develop cerebrovascular disease."

Dr. Adams and his colleagues, Dr. McKie, Dr. Kathy McKie, assistant professor of cell and molecular biology, and Dr. Nichols, are "in the early phases everywhere" on the study to develop a method to predict the risk of stroke in sickle cell children using TCD and MRI technology, he said.

The researchers must determine normal intracranial vascular flow rates because this is the first study of its type on children.

"Once the normal rates have been established, we will study children in the same age range with anemia who do not have sickle cell disease," Dr. Adams said. "This is necessary since the condition of anemia may affect the blood flow rates." In the same manner, criteria will have to be established for children with known or probable abnormalities. Co-investigator Dr. A.Q. Fischer, assistant professor of neurology, will provide assistance in pediatric ultrasonology.

"When we have determined all these criteria, then we will begin screening our target population," Dr. Adams said.

The researchers, using TCD, will attempt to screen all the children seen at the MCG Sickle Cell Disease clinic over a four-year period. The clinic sees about 200 children in the target age group and, figuring some growth in the number seen, about 60 will be screened each year.

The researchers hope that during the five-year study, advances in software design for the computers that assist both the MRI and the TCD will allow more accurate imaging. This may allow the investigators to examine more closely the blood vessels for abnormalities.

Dr. Adams sees the study as one segment of a larger set of problems: the cause of stroke in sickle cell anemia, prevention of strokes and finally, why they occur in this population.

While these questions are not addressed in the current study, the investigators feel some of the information gained could be useful in future studies to devise therapies to prevent strokes in sickle cell children.

"At the end of the study, we hope to have developed a technique to detect children who are predisposed to stroke before they have it," Dr. Adams said. "Doing that is the first step to answering the other questions." ■

—John Donnelly

Causes and Cures

It surfaced with an uncontrollable tremor.

Betty J. King could not control her left arm.

"I thought it was just my nerves to start with," the Hayne City, Fla., woman said.

In one sense Mrs. King was right. She has Parkinson's disease, a disorder affecting the portion of the brain involved in basic functions such as walking, facial expressions, balance and automatic speech.

Communication between the nerve cells in this portion of her brain had begun to break down. That's why the tremors had begun. It was a sign that something was amiss.

The brain of a Parkinson's patient lacks sufficient dopamine, a chemical that stimulates communication between brain cells, said Dr. Kapil Sethi, director of the neurogeriatrics clinic at the Medical College of Georgia.

Although the specific cause of Parkinson's is unknown, it is clear that the lack of adequate dopamine goes hand-in-hand with the disease.

"We know that dopamine deficiency causes all the cardinal symptoms of Parkinson's disease," Dr. Sethi said. Postmortem studies show low amounts of dopamine in the brain tissue of Parkinson's patients.

"Also, a street drug called MPTP causes all the cardinal symptoms and signs of Parkinson's disease. This drug seems to destroy selectively the dopamine-bearing neurons of the brain," Dr. Sethi said.

Exactly how dopamine works in the brain is not known, but one function seems to be generating movement.

Parkinson's patients slowly lose the ability to move normally. There's a decrease in the natural swing of the arms while walking. Walking becomes more of a shuffle. All movement becomes slower and restricted. Handwriting becomes small and difficult to read. Sometimes there is memory loss and confusion. But, "under their mask, usually there is a very intelligent brain," Dr. Sethi said.

Dr. Sethi in clinic with patient Lizzie M. Freeman.

A clinical examination is the primary method for identifying Parkinson's. No laboratory test exists.

Once properly diagnosed, patients can be given drugs that duplicate the work of dopamine. The drug levodopa, which converts to dopamine in the brain, helps compensate for the loss of dopamine experienced in Parkinson's disease.

The commonly-prescribed anti-parkinsonian agent is most effective in the first two to five years of use. Later, the drug becomes less effective in controlling symptoms of Parkinson's.

Drugs such as bromocriptine, which helps stimulate dopamine receptors in the brain, also are effective in reversing symptoms of the disease.

Sometimes surgery is performed to destroy an area of the brain where abnormal impulses originate. The surgery may help reduce tremors associated with Parkinson's.

Supportive measures, such as a loving spouse, also are extremely important for people with Parkinson's. Support is so important, in fact, that Dr. Sethi wants to establish a support group for Parkinson's patients and their families.

Mrs. King is one of the patients who come to the weekly neurogeriatrics clinic at MCG for treatment of a variety of neurological diseases that restrict movement, such as Parkinson's, or too much movement, such as Huntington's chorea. Patients with Alzheimer's disease, stroke and dementia also are treated at the clinic.

Dr. Sethi follows 50 to 100 patients with movement disorders. Some of the disorders are drug-induced, the unwanted effects of long-term use of drugs such as Reglan, a commonly-prescribed treatment for upper gastrointestinal problems like vomiting and nausea.

"You look at their charts and see people have been on Reglan for years," Dr. Sethi said. "You can do something there — remove the offending agent (Reglan)."

Other drugs such as Haldol are used effectively to treat severe psychiatric problems but produce involuntary movements. "These drugs have done wonders, but there is a price," Dr. Sethi said.

When the drugs are necessary to control a major psychiatric disorder, it means leaving the patient on the drug and looking for other measures to minimize side effects.

But sometimes Dr. Sethi finds patients on powerful neuroleptics for problems such as mild depression. Often these drugs are precribed too frequently for too long in too high a dosage, he said. The answer may be modifying the drug regimen, but sometimes damage is irreversible.

However it's Parkinson's disease that Dr. Sethi sees most commonly.

Levodopa effectively controls symptoms, especially initially, although it does not stop progression of the disease. But life expectancy is enhanced through the use of this and other drugs and treatment.

"I think it would be reasonable to say that well-treated Parkinson's disease patients will die of the same diseases his counterparts die of at that age," Dr. Sethi said. And, with proper treatment, "quality of life is so much better you can't imagine," Dr. Sethi said.

But there is much to do.

Despite evidence that dopamine levels are low in patients with Parkinson's and despite the obvious benefit of giving a synthetic dopamine, the cause of the disease is unknown.

Levodopa helps compensate for the loss of dopamine experienced in Parkinson's disease. But some movement disorders are caused by the very drugs prescribed to cure other ills.

A not-so-simple case of nerves brought Betty King to Dr. Kapil Sethi.

Environmental toxins have long been suspect. But everyone is expoxed to hundreds of toxins each day.

A Canadian study found that people who used pesticides or those who lived in rural areas where these were used had a much higher incidence of Parkinson's disease. The Canadian study also pointed to areas near paper mills as another high incidence area for Parkinson's.

But a Medical College of Georgia study comparing Georgia paper mills and rural areas with more urban areas found no difference in the incidence of Parkinson's.

"We have not been able to prove the hypothesis that pesticides in rural areas or paper mills have anything to do with Parkinson's disease," Dr. Sethi said. "But we cannot exclude the possibility that something else in the environment is causing the problem."

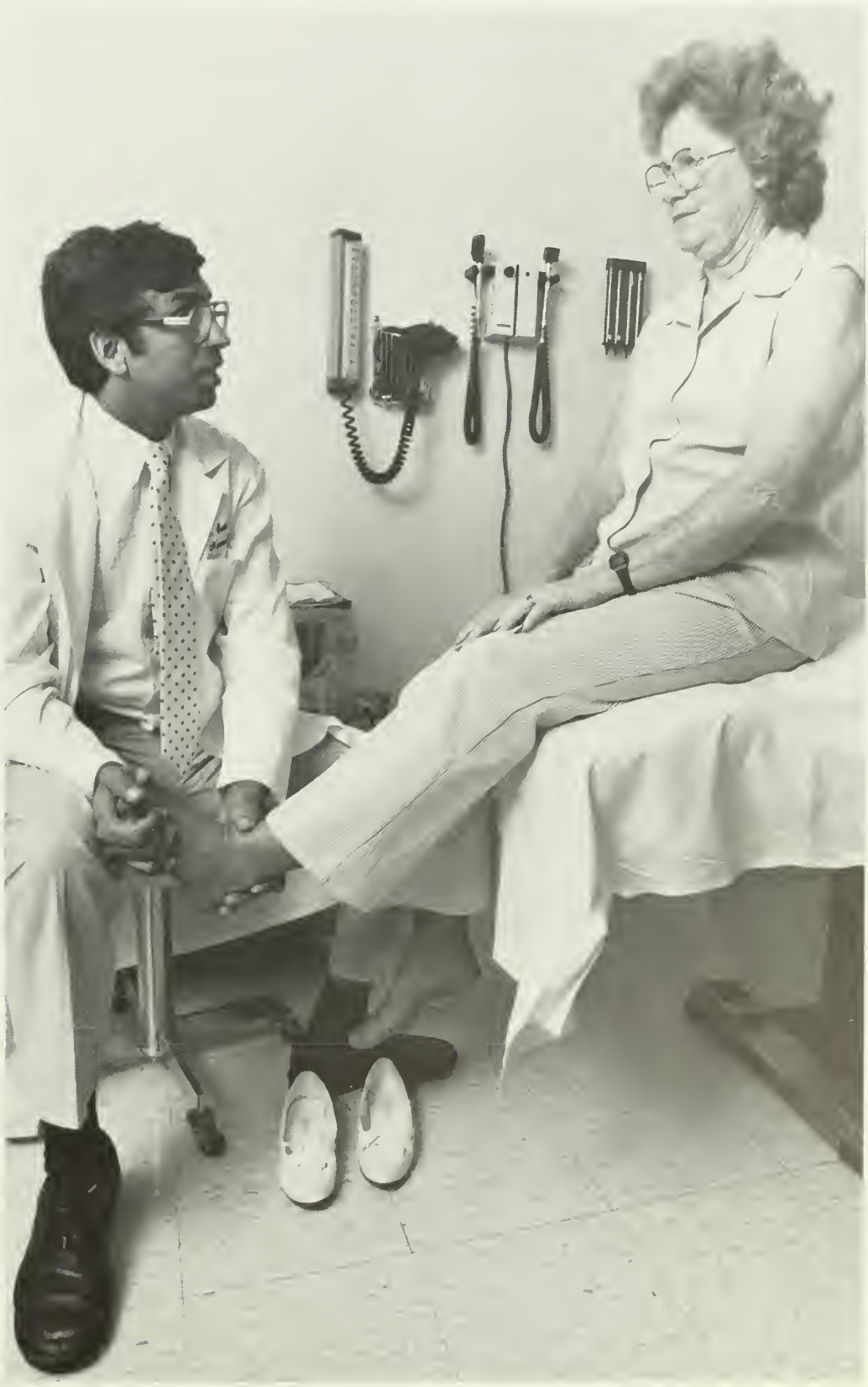
An area to be explored at MCG is development of a diet to maximize the effectiveness of levodopa for Parkinson's disease patients.

Levodopa is absorbed by the body just as amino acids are absorbed. Proteins are made up of amino acids and it's known that protein intake interferes with the absorption of levodopa and therefore its effectiveness.

Yet patients placed on standard low-protein diets tend to lose weight and muscle mass.

Dr. Sethi wants to devise a diet that allows for the maximum use of levodopa without causing unwanted weight and muscle loss. ■

— Toni Baker



An Insidious Disease

The Multiple Sclerosis Clinic aims to help its patients achieve independence, says its director.

The patient — brunette, petite, middle-aged — managed a fatigued smile. The sort of smile worn by those of whom life has asked too much.

"The wheelchair is deceiving," she told Dr. Daniel S. Feldman, director of the Medical College of Georgia Multiple Sclerosis Clinic. "I can get around without it."

The distinction that the wheelchair is for convenience instead of necessity was one she made several times during the doctor's appointment, and one she reminds her family of often.

But she has no delusions. In the same breath that she fervently asserted her independence, she chronicled the latest evidence of her body's betrayal. Her legs are increasingly weak and rubbery; her gait is shaky; one of her eyes throbs and robs her of the pleasure of reading. The simplest daily tasks are all she can manage, and the hours of television that occupy her time leave her depressed.

"I used to sew clothes for the family — even my husband and son," she said, adding that her unsteady hands have practically eliminated the activity. "I did make a pair of shorts this summer, but it took me two months." In better days, it would've been a matter of hours.

Such are the maladies of multiple sclerosis, a disorder of the central nervous system. The disease is one in which myelin, a substance which insulates nerve fibers, disappears. The cause is unknown, and the condition is incurable.

"Why the body chooses to make myelin disappear, we don't know," said Dr. Feldman. "But we suspect it has to do with the person becoming 'allergic' to myelin."

The disease, which usually strikes people in their 20s or 30s and affects more women than men, tends to be episodic. A series of attacks followed by partial or complete recovery is typical, though remissions may cease as scars replace myelin.

Symptoms vary according to the area of the nervous system affected, but may include weakness, tingling, numbness, impaired sensation, lack of coordination, disturbances in equilibrium, double vision, rapid eye movement, slurred speech, tremors, stiffness, paralysis or impaired bladder and bowel function.

The disease usually doesn't significantly affect life expectancy, according to Dr. Feldman. "Any chronic illness can shorten life expectancy, but it doesn't have to be severe shortening," he said.

And Dr. Feldman's goal for his patients is to help them live life to the fullest. The clinic, supported by the Georgia chapter of the National Multiple Sclerosis Society, has about 180 patients, all of whom are counseled to make the most of their abilities and to minimize their disabilities.

"The most traumatic thing about MS is the realization by the patient that he has it," Dr. Feldman said. "Wishing it away is not going to occur. If you make this adjustment, you can lead a happy life."

Dr. Feldman's goal for his patients with multiple sclerosis is to help them live life to the fullest.

Most of his patients do indeed lead well-adjusted, independent lives, he said, reeling off a list of some who have excelled in their private or professional lives.

"My goal is to assist the patient to develop an independent, adult existence," he said. "We each have a role to play in life. The role of adults is to be independent but interactive."

Nevertheless, the disease takes its toll, especially when severe symptoms such as paralysis occur. Some patients forgo having children, and others watch their marriages dissolve.

"This is a tremendous trauma to a marriage," Dr. Feldman said. "I always admire supportive family members."

Aside from support, what can the medical community offer MS patients? Quite a bit, according to Dr. Feldman. Physical therapists help patients realize their physical potentials. Dr. Feldman conducts extensive counseling sessions with patients in which such things as diet, exercise, lifestyle and depression are discussed. Certain hormones or steroids, thought to lessen the intensity and shorten the duration of symptoms, also may be prescribed.

And more help is on the horizon. Although a cure is not imminent, Dr. Feldman said research is refining medications to increase remission periods, thus minimizing damage to the nervous system. ■

—Christine Deriso

Mission of Nursing

Carolyn Gaffney is a member of Paralyzed Veterans of America — because of her devotion to spinal care nursing.

Consider the victim of a spinal cord injury: In a split second, he may be transformed from a state of health and vitality to one of immobility, dependency, helplessness.

The consequences present an enormous challenge to the health care worker who will spend more time than any other in his rehabilitation: his nurse.

Those consequences are both physical and emotional. Rage, hostility and depression are common reactions to the paralysis that may result, and nurses often are the target of these feelings. This, coupled with the fact that most spinal cord patients spend months in the hospital, makes their nurses seem particularly worthy of the title, "angel of mercy."

"It's rehab, and not everybody likes rehab nursing. But it's a very rewarding experience," said Carolyn Gaffney, a nurse at the Veterans Administration Medical Center who has devoted a lot of energy in the last four years to recruiting nurses to the field.

Mrs. Gaffney received a master's degree in adult nursing from the Medical College of Georgia in 1981 and embarked on her recruiting mission with particular zeal. Nursing was a dream she'd harbored since childhood, but she didn't make it a reality until middle age.

"I had an aunt who was a nurse, and I just idolized her," she said. "She looked like an angel to me." But Mrs. Gaffney's life took a different course when she married a man in the Navy. Her life then consisted of traveling with him, raising their three children and pursuing a career as a fashion model.

At 40, she was ready for a fresh start. Recently divorced, she recaptured her old dream and enrolled in the MCG School of Nursing. While earning her master's degree, she worked with spinal cord patients and decided to pursue it after graduation. She soon was made head nurse of the Veterans Administration Medical Center's spinal cord unit, and fell in love with the emotional payoffs.

"If it's nothing but being able to move a head or arm, it's so rewarding when they improve," she said.

Her enthusiasm prompted her to join the Paralyzed Veterans of America, a national organization. She's been a member for five years and is chairman of the organization's nursing scholarship program.

"We were talking about the shortage of nurses on the spinal cord unit during a meeting, and thought if we offered scholarships, we could attract some," she said.

In the four years since the program began, the organization has awarded eight scholarships, which pay everything but room and board. Recipients agree to devote one year to spinal cord nursing for every year they receive financial help.

"(Scholarship recipients) have to be in need and come from a family who can't afford to send them to school," Mrs. Gaffney said. Mrs. Gaffney interviews them, reviews their grades and makes recommendations to the organization. She has plenty of potential recipients to choose from.

"All during the year, I get calls about it," she said. "Word has gotten out that it's a good program."

Mrs. Gaffney is so pleased with the program that she has stuck with it even though she's moved on to a series of new challenges. For one thing, she recently remarried. For another, she's begun a new nursing position with the MCG Comprehensive Diabetes Care Center. But her love of spinal care nursing will never wane.

"I still see spinal cord patients, and one day, I'll probably go back to it," she said. ■

— Christine Deriso

Carolyn Gaffney visits a spinal cord patient at the VA Medical Center.

Nerve-Muscle Link in Amyotrophic Lateral Sclerosis

Medicine knows little about the killer amyotrophic lateral sclerosis, ALS.

The little that is known is bad.

Nerves and muscles die, turning arms and legs into useless pipe stems. Victims lose the ability to talk or even swallow. Ironically the heart seems unaffected and often the mind remains intact. Eventually breathing becomes impossible. Death is almost inevitable.

The highest incidence is in men in their 50s and 60s. Yet the disease can strike at random, hitting young men and women alike.

Five percent of the time the neurological killer runs in families. Usually it occurs at random.

"If you were going to invent a more diabolical disease, it would be hard to do," says Dr. Thomas R. Swift, chairman of the Department of Neurology at the Medical College of Georgia.

Baseball player Lou Gehrig had the disease, hence its popular name. Anywhere from four to seven people per 100,000 have ALS. The prevalence rate is relatively low because victims typically die within three years of diagnosis.

The how's and why's of the disease are unknowns.

"Everyone is looking for the cause," Dr. Swift said of the flurry of research activity that has built around this killer.

Many causes have been proposed, anything from environmental toxins to heavy metals to immunologic attacks, said Dr. Michael H. Rivner, MCG neurologist.

But nothing has been proven.

"Neurons die with aging. That is known," said Dr. Adarsh K. Gulati, assistant professor of anatomy at MCG. "It may be that ALS is due to a faster aging process."

Doctors at the Medical College of Georgia are working to fill in some of the blanks about ALS.

Drs. Gulati, Swift and Rivner are looking at what may be at the heart of ALS. They are examining the interaction between nerves and muscle.

"We are trying to find the

Studying muscle transplanted from ALS patients into nude mice, Drs. Gulati, Swift and Rivner seek to understand the interaction of nerves and muscle in ALS.

Researchers hope experimenting with diseased and normal tissue will help them understand the relationship of nerves, muscle and ALS.

Researchers find that diseased human muscle becomes normal again once innervated with healthy mouse nerves, such as the one exposed here.

indirect relationship between the nerve and the muscle and how it is disturbed in ALS," said Dr. Swift, who took a six-month sabbatical from his role as chairman to do ALS research with Dr. Gulati.

What they have found so far does not surprise them.

The Medical College of Georgia doctors have taken diseased muscle from ALS patients and transplanted it into mice without immune systems.

But once the mice's nerves worked their way through the muscle, the muscle became normal again.

"What that tells us is that the primary lesion or cause is the nervous system," said Dr. Gulati.

Both muscle and nerve have been blamed over the years for the destruction caused by ALS.

"Most people believe it's a disease of the nerve, not muscle," Dr. Rivner said. "The fact that the muscle looked better when you put healthy nerve into it is not that surprising."

It also has been proposed that ALS is caused by a primary abnormality in the muscle that was somehow killing nerve cells, Dr. Swift said.

That theory had not been disproven. But "this is evidence against that theory," he said.

Dr. Gulati says the work is important from two perspectives.

The ability to transplant ALS muscle successfully into nude mice is a promising model both for ALS research and for the study of other neurological problems.

Also, the fact that the diseased muscle worked and appeared normal after re-innervation supports the belief that the nerve may set the deadly ALS cycle in motion.

But the researchers are not satisfied. They have applied for National Institutes of Health funding to continue their studies.

It takes eight to 10 weeks for the mice nerves to infiltrate the ALS muscle and reverse muscle atrophy. The MCG project has followed mouse models for a maximum of five months. During that period, there has been no muscle change to indicate the disease was resurfacing.

People with ALS typically get the disease later in life, Dr. Swift said. But it may be that the muscle has been poisoning the nerve for years.

That's why the researchers now want to study the muscle for longer periods to see if any changes occur. A mouse lives two years. The MCG doctors would like to study the muscle up to one year after the transplant to ensure that no changes occur to implicate the muscle. That year in a mouse's life corresponds to about 55 years of human life.

"If ALS muscle is killing nerves, it should kill the nerves in the nude mice," Dr. Swift said. "That is why we are continuing to see if we can find any evidence that it is killing the mouse nerves."

And they want to take still another approach.

The doctors plan to make an extract from diseased human brain tissue and put it in tissue culture with normal motor neurons from rats or mice.

The idea is to see if the diseased brain extract will affect the normal neurons, Dr. Gulati said.

"We anticipate that it may," he said.

They also will give the brain extract to rats and mice to see if they begin to develop ALS.

The researchers hope the disease occurs in the rodents and provides a model for studying the disease and finding a way to fight it.

"The two key components involved in this disease are the nerves and the muscle," Dr. Gulati said. "By doing these combinations of diseased and normal tissues we should be able to find out which is more involved in causing ALS." ■

— Toni Baker

Living with Dignity and Courage

Mary Jo Fitzgerald stumbled over her own two feet.

She first stumbled on the unfamiliar turf of a tennis court.

Mary Jo laughed. Everyone laughed.

It was 1984.

Life was good for Mary Jo and Bob Fitzgerald. They loved each other and their six children.

Weeks later, they were vacationing in Bermuda.

Mary Jo was soloing on a moped. She hit a parked car and went over the moped's handlebars. She seemed fine, but her leg began to spasm.

Everyone was still laughing — it didn't seem important.

Then one day she stepped out of the family van and her legs gave way.

No one was laughing, but no one was really scared either.

This was probably some virus, they reasoned. How could anything be really wrong with Mary Jo?

She was vivacious. She was strong. She set tough, high standards for herself and her family. She was passionate and pioneering.

But something was very wrong. The first neurologist was not sure what.

The second one put her through more tests. By then Mary Jo had done her homework. She asked the second neurologist if she had amyotrophic lateral sclerosis, ALS.

He was shocked at the question.

The couple was stunned by the answer.

A muscle biopsy confirmed their suspicions.

"You don't do anything," said Bob Fitzgerald. "You sit there in an incredible sense of helplessness. All you are faced with is one hard, cold fact that you can't deal with, you can't correct. Problems are nice, because problems have solutions. If something doesn't have a solution, you've got a fact."

The fact made the couple cry.

Mary Jo said something must be done. The neurologist told her there was nothing. She insisted. But there was no real hope he could offer.

They wanted a second opinion.

"All the hurt is there," Bob said. "You learn very quickly that hurt is one of two things. It's destructive if you let it be destructive or it's redemptive if you let it be redemptive. You feel small, you feel weak and you recognize that you are. But there is a human spiritual strength that becomes very, very apparent."

Bob Fitzgerald doesn't believe it when people say they don't believe in some higher being.

"There is a sustaining force there that will come and tell you right then and there, don't worry about it. This is part of a plan."

Driving home, Mary Jo said something that underlined her intention to live with this catastrophe as she had lived her life.

Bob smiles when he remembers that moment.

"'What we must do now is to live a life of courage for the children. That is my legacy,'" she told him. Her other comment was a little peculiar. Mary Jo told her husband that he would have to remarry.

"I said, you know we have been married for 20 years and you have done a great job of being a part of my life during those 20 years. But I will be damned if this isn't going too far."

And so began the story of an Atlanta family's experience with the deadly neurological predator, ALS.

The search for a second opinion brought Bob and Mary Jo to the Medical College of Georgia.

Dr. Thomas R. Swift, chairman of the MCG Department of Neurology, had no better news for them.

As a physician and as a man, Dr. Swift had lived out this scenario too many times.

He just completed a six-month research sabbatical looking into the cause of ALS.

"I got tired of seeing new patients every month and reading about their funerals and seeing them go downhill and feeling like I wasn't doing anything to try to find the cause of it," Dr. Swift said.

But he could do something for the Fitzgeralds.

Bob Fitzgerald

Mary Jo Fitzgerald has been living with a death sentence for three years. But it has not daunted her will to live, says her husband.

Bob Fitzgerald, flanked by photographs of his wife and children.

He gave them support.

"I don't really think he realizes the incredible impact he had on Mary Jo in the sense of courage and purpose he passed on to her," said Bob. "He was so willing to share information in a very realistic way, but was so obviously attuned to the hurt and human degradation of this disease."

So Mary Jo and Bob went back to Atlanta with the knowledge that the relentless disease was a fact of their lives and, short of some miracle, would be the death of Mary Jo.

The couple gathered their children, Kelleen, Maureen, Robert, Kista, Kerry and Meghan.

"Mary Jo was crying, not sobbing, but tears were flowing," Bob recalled. "I said, 'We have something we need to tell you. You know Mother and I have been going to the doctor.' That was just about as far as I got. When Mary Jo finally sort of got it out, I came back in, and she and I told them together."

From the very beginning, each of the children handled the situation as the individuals they are. The oldest asked just one question. The next child, her mother incarnate, asked a thousand questions. Their only son took it all in. Another teenager asked a few questions and cried.

Kerry, now 8, was 5 at the time. She cried because she knew something was wrong, but she did not understand what. Finally, she stood up, put her hands over her ears and said, "Enough, enough, enough."

"We all agreed when this thing first came up that it was going to be okay to cry and that we were going to touch each other and hold each other and it's okay to yell at each other," Bob said. "But if you are going to yell at somebody, you might get yelled at back. That was okay, too."

It also was okay, at times, to be sad and angry. It was okay, at times, not to want to be with Mary Jo.

Three years have passed since those first anxious moments when the diagnosis was made.

Mary Jo has gone from one cane to two canes to a walker to a wheelchair to a bed.

She communicates through an alphabet board. She is nourished through a tube in her stomach; still, the loss of muscle makes her appear malnourished. A nurse is now in the Fitzgerald house around the clock. Someone cooks the meals and cleans the large house.

But Mary Jo has not really changed, says her husband.

She made the concessions unwillingly, holding onto the walker as long as she could, refusing the stomach tube until she scared herself by choking on food.

When she can no longer breathe on her own, Mary Jo says she will not be put on a ventilator.

Bob describes his wife as passionate, but not demonstrably so. He knows her tenderness and that now causes him pain.

But the children have not really seen the tenderness. That can and does cause problems.

"The children intellectually understand the incredible depth of their mother's love. She is always there," Bob said. "They haven't always seen it demonstrated in a tender way. They see it demonstrated in the incredible price Mary Jo has paid in setting standards and seeing that they are abided by. They have seen it now in the incredible courage she lives with and her beautifully human inability to give up that maternal role." To this day, Mary Jo still lets her children know if they are leaving home inappropriately dressed.

Because Mary Jo's spirit has not left the body that betrays her, her spirit has moved others.

A lawyer friend had a client who had embezzled money and faced a jail sentence. The client said his family couldn't take the disgrace. He was going to kill himself. Their lawyer friend shared Mary Jo's story with his client. The client cried. He is now serving his

time. The Fitzgerald family has another friend who was mistreated as a child. She wrote Mary Jo that the peace and love she saw in Mary Jo helped her find peace.

"Sometimes I lose control of it all," Bob said. He worries about not being involved enough with the children. He worries about not being at work enough as vice president and general counsel of Southern Bell. At times, he must get away from it all. "I have told Mary Jo, 'Stop, my total system is overloaded and if you give me one more piece of crappy information to put into it, I am going to explode and I walk away from the situation.'" An hour later he is back.

Sometimes he just wishes everything was done and he has talked with Mary Jo about that. He knows some things will be easier when Mary Jo is gone. He believes that she is ready to die.

"It's not a wish that Mary Jo was dead. It is a wish that our life was different and a wish that life was easier. It's not going to be easier (when she dies) for who I am and who the kids are. The emotional hurt is not going to be easier."

This is where Bob turns to his strong faith.

"I don't look upon this as an absolute, total negative," he said. "I don't look upon it as a negative to have Mary Jo be the Lord's instrument in dealing (with others' problems). I don't look at it as a negative that our children will never be what they would have been otherwise. I don't even know what that is. I will never know what that is. They are going to be different, but if I am right in my basic premise that it is either going to be destructive or redemptive, and we can make it redemptive, then it is going to be better than it would have been otherwise.

"We are still hurting. But let me tell you, there is an incredible amount of hurt everywhere.

"We don't have an abnormal life. We have an intensified life. We are still dealing day to day with the same things everybody is dealing with. I go home and I have a loving wife at home. She's there in a wheelchair or a bed, but she is just as loving. She is just as much a part of my life as anybody's wife is. It's just an intensified loving."

So the family lives and loves and struggles sometimes.

Talk about what the future holds and Bob Fitzgerald will say "if" Mary Jo dies.

"Am I holding out for a miracle? Sure, that would be great," he said.

But he does not live in what he calls a spiritual fool's paradise.

"Basically whatever is going to happen is out of my control, is out of Mary Jo's control. We don't have a disease that we know how to cure. Nature and the Good Lord's plan are going to be carried out.

"He may decide that He needs to use her once more, for the object of a miracle," Bob said. "I don't hold onto that. It doesn't give me strength for the occasion. It gives me strength in a relationship with the Lord because it gives me strength in saying if it doesn't happen, that is the best thing, too. My faith says that if she dies, she died at the right time.

"Mary Jo will welcome death. Death isn't hard. It's the living that is tough. But as long as we have the gift of life, it is worth living with dignity and courage. Mary Jo is doing that." ■

— Toni Baker

Seeking Embryonic Clues

Dr. Gurkirpal Sohal hopes to discover clues to the causes of ALS by studying the life and death of nerve cells.

Dr. Gurkirpal Sohal has nerves on his mind.

"The fundamental issue with amyotrophic lateral sclerosis patients is what makes the neurons live and what makes the neurons die," said the Medical College of Georgia professor of anatomy.

"If we knew for sure what makes them live or die, we should be able to prevent them from dying. So, you need a system to study what factors operate on the nerve cell which makes this decision that this cell will die and that one will live," Dr. Sohal said.

He has found a model for studying the life and death of nerve cells in the embryos of chickens, ducks and quail.

In the developing embryos of these birds and even human beings, the nervous system produces twice as many cells as are needed, meaning about half of the nerve cells die.

Within these embryos is a naturally occurring situation where some cells live and others die, Dr. Sohal said.

"That's what we have in ALS. We thought we would use embryos as a model to study factors which control the survival of (cells) using chicken, quail and duck embryos as a

The life and death of a nerve cell may provide clues to understanding the destruction caused by ALS.

model."

These embryos also develop fairly quickly; a chicken hatches in three weeks. Eggs also are inexpensive and embryonic systems are simpler to study than adult brain tissue.

Dr. Sohal already has used embryo models to begin to unravel the many mysteries of ALS.

His research is as complicated as the body process it involves.

The body's central nervous system comprises the brain and spinal cord. The peripheral nervous system comprises the body's network of nerves.

For movement such as raising the arm to occur, the nerve cells in the central nervous system must communicate with the nerves in the peripheral system which in turn communicate with the muscles.

That system is somehow destroyed by ALS, and nerves and muscles die. But medicine does not understand what causes this destruction.

"People used to believe that the muscle provides factor, (the) chemicals which make the neuron live," Dr. Sohal said. "Some of the experiments that we have done tell us that the factor that we are looking for may not be coming from the muscle, but from the brain itself."

"Assume for argument's sake that the half of the neurons that die in the embryo die because only so much of this factor is in the muscle. In other words, the muscle is too small to produce enough factor for all the neurons. How do you test that? What you do is reduce the number of neurons in the brain," Dr. Sohal said.

If the number of neurons is reduced to the level that normally survives, then no further neurons should die, he said. There should be ample factor in the muscle to support the neurons, if, in fact, muscle is the source of factor.

Dr. Sohal knew that the duck's brain is twice as big as the quail's brain. It seemed logical that if he transplanted the brain of a quail into the duck embryo, the duck embryo should have ample muscle factor to support all of the quail neurons. No neurons should have died.

But they did die.

"That tells us (the factor) is not in the muscle," Dr. Sohal said.

So he took the opposite approach.

He left the duck brain intact but moved muscle from the quail to the duck. That should have meant that the duck had less muscle and that more than the normal number of neurons should die.

Only the normal number of cells died.

"That tells us that the size of the muscle is not correlated with the number of neurons surviving," the researcher said.

He documented the findings and has published this work.

Over the next year, Dr. Sohal and his laboratory staff will examine the role of central connections in nerve cell survival.

Central connections are the means by which nerve cells in the central nervous system control nerves in the peripheral system.

Dr. Sohal wants to alter the number of nerve cells involved in this central communication and observe the effect on the number of surviving nerve cells.

If he reduces the number of nerve cells communicating with the peripheral nerves, more of those peripheral nerve cells should die. If he increases the number, more should live.

His objective is to document that the destruction caused by ALS begins in the nerve cells. The second issue is what factor in the nerve cells causes the problem.

"If we can see differences as a result of manipulations, then the next step would be to find out what in the nerve cell is causing the problem," he said. "In science, what you do is go with what you have."

Today Dr. Sohal believes he knows that ALS originates in the nerve cell. But he knows that is not an absolute.

"The only way we can find out is by doing more and more research. Some cells die every day. Most organs regenerate and repair damage. In the central nervous system, regeneration does not occur."

Dr. Sohal hopes to identify the factors operating for the survival or death of nerves. Should that factor be some chemical or a mixture of chemicals, it might be possible to give the chemical or chemicals to ALS patients if they lack sufficient amounts. He likened the process to giving insulin to a diabetic.

His work to date has been funded by the National Institutes of Health, and he recently secured a five-year grant from NIH to continue these and other studies.

Drs. Rebecca Campbell, Iftekhar Husain and Rudy Ravindra work with Dr. Sohal in his lab.

Bird embryos provide a model for Dr. Sohal's experiments with nerve cells.

New Lines of Communications

And their interest in the nerves does not stop here.

Research also is underway to understand synapses, points of communication between two nerves or a nerve and a muscle cell. All nervous system communication occurs through synapses.

To study this important juncture, Dr. Sohal is using the neuromuscular synapses because they are larger than the synapses between two nerve cells. For this project he also is using chicken, quail and duck embryos.

"Normally, people think that the nerves go to the muscles and tell the muscles that we want to make a synapse here and the muscle simply obeys," Dr. Sohal said. "We find that that view is wrong."

Rather, Dr. Sohal has found that the muscle decides where the synapse will occur.

"We wanted to know what the muscle could do if the nerve was not there," he said.

To observe this, he needed a system in which he could ensure that the nerve never reached the muscle.

He was able to do this in eggs by destroying a part of the brain which controls the nerve-muscle communication. He then studied the muscle to see if specializations occur that indicate preparation for a synapse.

He found that the muscle does select and prepare a site for a synapse.

"We were looking at the same muscle and correlating the number of nerves going to the muscle and how many synapses the muscle had. If the nerves were inducing synapses, then every time you have more nerves in an animal you should have more synapses," he said.

Ducks, which have the bigger brain, should have the most synapses; chickens should have slightly fewer, and quail should have the least.

"It didn't work quite that way," Dr. Sohal said.

The work at MCG showed that the muscle controls the synapse.

"You need to know everything you can know about these two ends, the end connecting with the muscle and the central connections, because these are the only two places where cells communicate," Dr. Sohal said. Through these junctures, information moves from the brain to the nerves and muscles.

Understanding these important communication methods can lead to better understanding and, possibly, better treatment of a variety of neurological diseases.

Now that he knows nerves are not necessary to prepare for synapses, the question remains: what is necessary? The connective tissue in muscle may play an important role. But how does the muscle know where the synapse needs to be? It appears from previous work that the muscle typically forms the synapse in the middle of the muscle. The question remains: why there?

At this point, Dr. Sohal has begun to destroy muscle to see if the nerves form a synapse. So far, it appears that nerves without muscle do not prepare for synapse.

"I think there is something here which is telling us that there is something which probably does both things, that is making the nerve specialize and the muscle specialize," he said.

Whatever that one thing is, Dr. Sohal believes it may be found in the muscle. ■

— Toni Baker

Dr. Adarsh K. Gulati is building biological bridges.

He wants the bridge to take him from a nerve that is irreparably damaged to a nerve that functions once again.

"As we know, nerves can be injured a number of different ways," said the assistant professor of anatomy at the Medical College of Georgia.

Crushing injuries, gunshot wounds, blood flow problems all cause nerve damage.

Sometimes the nerve repairs itself. But many times the injury irreparably damages the nerve and interrupts vital communication that begins in the brain.

An injured arm or leg may be salvaged by surgeons but may be useless to the patient because it no longer has any feeling or movement.

The brain gives orders to the neurons or nerve cells in the body which, in turn, innervate the muscle so it can perform some activity. Neurons or nerve cells control muscle activity. The neurons in the brain are upper motor neurons and control the lower motor neurons in the spinal cord. Those, in turn, control the muscle.

"It's a hierarchy-type system," Dr. Gulati said.

Axons, or nerve fibers, come from the lower motor neurons and form neuromuscular junctions.

But say there is a peripheral nerve injury from trauma. If the injury is minor, the axons regenerate and re-innervate the muscle.

In other cases, if remaining nerves are close enough together, neurosurgeons suture them, and the axon can grow again.

But sometimes the gap is too large for either effort.

Then neurosurgeons may take a less important nerve from another part of the body to bridge this gap. That means damaging that nerve. Also, if the nerve graft is not as large as the damaged nerve, function of the nerve will never completely recover.

"The best type of graft or material you can use is a nerve of the same size to bridge the gap," Dr. Gulati said. "The only way you can get that graft is from another individual from the same nerve."

Frozen nerve banks have been recommended to meet this need, and nerve grafts from frozen nerves have been used. But results to date have been less than ideal. Often, regeneration does not occur.

"Nobody knows what this ideal material is which you can use to bridge long gaps," Dr. Gulati said.

Using rats as a model, Dr. Gulati has created nerve lesions and tried various methods of bridging the gap.

"What we have shown is that if you take a graft from another animal, it gets rejected because the host would create an immune response which would reject the graft," he said.

He tried immunosuppression, similar to the methods used in vital organ transplants, and found that immunosuppressants help axons grow through the long grafts of the peripheral nerves.

But as soon as the immunosuppressants are stopped, the rat's body begins to reject the graft, and axons degenerate and die.

An option could be lifelong maintenance of immunosuppression, but Dr. Gulati does not believe that is a reasonable alternative considering potential side effects.

He looked further to identify

Experimenting with nerve grafts is yielding some promising information about healing injured nerves.

Dr. Adarsh Gulati uses rat models to experiment with grafts to repair injured nerves.

the component of the nerve graft essential for regeneration and found the basal lamina and schwann cells to be two important components of the graft.

Basal lamina alone can support regeneration of the axons. Schwann cells within the nerve are involved in eliciting the body's immune response. Fortunately, schwann cells can be destroyed by freezing and thawing.

Another substance in the graft is called myelin. When a nerve is cut, myelin becomes debris and can interfere with the growth of axons through the graft.

Dr. Gulati found that if he injured the donor nerve and waited four weeks, the myelin degenerated and was taken up by the schwann cells. When the graft was frozen and thawed, the schwann cells also died. Using certain culture and other techniques, Dr. Gulati then removed the dead cells from the graft.

"What we are left with is an acellular basal lamina nerve graft," the researcher said.

"You can hypothesize since they do not have any cells in them, they are going to be less immunogenic and better tolerated by the hosts," he said.

Preliminary work has shown that the grafts do have some immunogenicity, but much less than do grafts containing cells.

Now, Dr. Gulati wants to use these acellular basal lamina grafts in rats to see how well the axons grow and whether the process can be used first experimentally and, eventually, clinically to repair gaps in injured nerves.

He believes it may still be necessary to use some immunosuppression with these new acellular grafts, but probably only for a short time. Short-term use of the drugs could be justified, Dr. Gulati said.

Should the animal research prove successful, it would be possible to prepare and use acellular basal lamina nerve grafts for human nerve repair, Dr. Gulati said.

The Medical College of Georgia researcher already has begun some of this work and recently was funded by the National Institutes of Health to pursue the study further.

Should this approach to nerve grafts prove adequate in the peripheral nervous system, it may work in the central nervous system, an area where nerve regeneration has been thought impossible, the researcher said. ■

— Toni Baker

Relief for TMJ Sufferers

Linda Hastings' headaches were so intense she sometimes felt her head would explode.

Since 1977 she had suffered constant, debilitating headaches. In search of relief, she saw numerous health professionals — from a neurologist to a psychologist — and tried therapy that ranged from biofeedback to anti-migraine medications.

About a year ago, a doctor referred her to the Craniomandibular Pain Clinic at the Medical College of Georgia School of Dentistry, and Mrs. Hastings found relief.

At the clinic, MCG professor Dr. Gerald H. Loft delved into Mrs. Hastings' history. He asked questions about her headaches, examined her jaw muscles and evaluated her occlusion, or bite, to see if an occlusal imbalance might be causing the pain.

After his assessment, Dr. Loft's prescribed treatment was a plastic occlusal balancing splint worn over Mrs. Hastings' maxillary teeth to correct her misaligned bite. His diagnosis was that her headaches stemmed from muscle spasms related to a craniomandibular misalignment and stress. She was instructed to wear the splint 24 hours a day, except when eating.

Mrs. Hastings didn't hesitate to wear the sometimes awkward splint. "I had no guarantees, but I had nothing to lose," she said. The pain of her headaches sometimes had sent her home from work, or to the emergency room for a shot of dilaudid, the only medication that seemed to help.

Six months later, Dr. Loft's diagnosis was confirmed. Mrs. Hastings no longer needed the anti-migraine medications she had been taking all those years. She was no longer in pain. The tension from her misalignment had been relieved.

Craniomandibular dysfunctions cause pain for many. And many patients with craniomandibular pain have seen five or six physicians and dentists before finding help, according to Drs. J. Marvin Reynolds, professor of prosthodontics, and James D. Allen, assistant professor of prosthodontics, both of whom work in the clinic.

Most of the patients who find their way to the clinic are there on referrals. Most suffer from chronic pain and have been seeking relief for several months. Many are women between ages 35 and 55, according to Dr. Reynolds.

TMJ, a popular term for craniomandibular disorders with a dental component, refers to temperomandibular joint syndrome. According to Dr. Reynolds, problems of patients they see in the clinic rarely have a single cause.

"The chronic craniomandibular pain problems that dentists deal with are either capsular or muscular in nature," said Dr. Reynolds. Capsular refers to the membranous covering around the temperomandibular joint which holds disc-like cartilage in place.

The most common cause of muscular dysfunction is bruxism (clenching the jaws) due to stress. The problem is often aggravated by an orthopedic imbalance to the temperomandibular joint due to an improper bite.

Capsular problems usually appear in some form of disc derangement which is frequently complicated by inflammation, Dr. Reynolds said.

Although the relationship between the head, jaw and muscles that complete the system are key contributors, there's another, possibly more important, component: stress.

In Mrs. Hastings' case, stress played a major role.

"At the time that I met Dr. Loft I was working two jobs and going to school," she said. Linda's life is still hectic. She's expecting her second child and still works two jobs, one at MCG as a computer operator in the clinical pathology lab and another for an Augusta gynecological clinic.

Stress can cause muscles to contract more than normal, lowering blood flow through the contracted muscle and leading to pain.

Clenching the teeth is one sign that the symptoms are stress related, and job dissatisfaction, family problems, life changes or other stressors can all play a part in the pain. Often, the stressors must be alleviated to stop the pain.

Clinic doctors often refer patients to MCG Dental Practice Dynamics to see Dr. Robert F. Kaltenbach, a psychologist, for counseling and/or biofeedback. At times, the patient must decide to make lifestyle changes or behavioral

Dr. Gerald Loft checks for tenderness in Linda Hastings' jaw muscles.

People with temporomandibular joint syndrome sometimes find their way to the Craniomandibular Pain Clinic by a circuitous route.

Dr. Loft tests for correct bite while splint is in place.

modifications.

"Sometimes it just comes down to the patient making a quality of life decision," said Dr. Allen. "They have to ask themselves how much the stress is affecting their life."

Craniomandibular problems can also stem from trauma, nutritional deficiencies and posture problems.

Along with the multiple components involved, pain usually occurs in more than one area. But one complaint stands out above the rest.

"There's no question about it," said Dr. Reynolds. "Of all complaints, headaches are by far the highest (incidence)." A dental occlusal imbalance may be a contributing factor in as many as half the cases involving chronic headaches.

Earaches, joint pain, joint noises, joint motion problems, jaw pain, facial pain and even toothaches are other complaints caused by muscle dysfunctions.

Diagnosis is no easy task, not only because so many symptoms may be involved but also because many other conditions can cause similar complaints and signs. Included are vascular problems, nervous system disorders, organic diseases, tumors and psychological disorders.

"We cannot have tunnel vision and think purely of dental factors," Dr. Reynolds said. "There could easily be one hundred possibilities for some complaints.

Getting a patient's history is the first step in the diagnosis process. A patient in the TMJ clinic must first fill out a screening questionnaire to give doctors a broad overview of his medical history and problems.

After the patient has completed the questionnaire, the doctor asks further questions. Since stress is often a factor, Dr. Reynolds tries to get an overview of the patient's emotional well-being.

From both the questionnaire and their questions, the doctors try to pinpoint the biggest contributor to the problem.

"We may narrow it down to five possibilities," said Dr. Reynolds. "Then we try to pinpoint which one plays the biggest role."

"We do the least invasive thing necessary," said Dr. Loft. "Initially, we want to do the least to get them well."

Often, the doctors begin with repositioning splints. Splint therapy serves both as an initial pain-relieving treatment and as confirmation of a diagnosis. Muscular problems due to occlusal imbalances should respond rapidly to such therapy. If the headaches or other symptoms persist, then they investigate other procedures.

To complement splint therapy and stress reduction, physical therapy is often used and the doctors often work closely with the MCG Physical Therapy Clinic.

"The more types of therapy that you instigate, the more success you'll have," Dr. Allen said.

After the signs and symptoms have been resolved through splint therapy and other procedures, the doctors make a thorough diagnosis of the malocclusion and outline appropriate corrective procedures. They might recommend irreversible procedures such as orthodontics, orthognathic (jaw aligning) surgery, tooth reshaping, selective grinding, prosthodontic replacements or restorative procedures.

The doctors hope to achieve a common goal through all their questioning, diagnosing and testing. In the end, the bottom line, according to Dr. Allen, is that the "treatment has to be directed at making the person's quality of life better." ■

—Karin G. Calloway

Bettering the Odds

Robert Shinault has grown up much like any other teenage boy. At 14, he has played baseball on his junior high school team and works as a busboy three nights a week. To look at him, you'd think he's an average high school freshman.

But the night of Robert's birth wasn't by any means average. His father, Curtis Shinault, remembers, "When he was first born, that was the scariest thing we've ever been through."

Along with other complications, Robert was born with a myelomeningocele, a congenital defect in which spinal membranes and cord protrude from the vertebral column. He underwent surgery to repair the defect shortly after birth. At three months, Robert began showing signs of hydrocephalus: fluid began to collect in the brain cavity. He immediately underwent surgery at the Medical College of Georgia Hospital. There, a shunt was implanted to divert the build-up of fluid from his head, along his neck and to his abdomen.

"We were scared to death and no one could really tell us anything about the rest of his life," Mr. Shinault said.

The fact that Robert lives a normal life would have been a miracle before the late 1950s. Before that time, hydrocephalic babies filled the wards of asylums. Without the benefits of shunts, their heads would often grow to nearly the size of a watermelon and the syndrome generally led to severe brain damage and retardation.

The problem of moving

Robert Shinault, 14, bears no ill effects from his experience with hydrocephalus as an infant.

trapped cerebrospinal fluid from the brain cavity and depositing it back into the bloodstream troubled doctors for years, said Dr. Marshall B. Allen Jr., chief of neurosurgery at MCG. Cerebrospinal fluid, which covers and protects the brain and spinal cord, follows a route up through the spinal column, around and through the brain and back through the spinal column.

Shunting a patient with the syndrome led to many complications.

"In the old days, tubing broke and caused clots when in the blood vessels and a reaction when in the abdomen" which would lead to blockage of the shunt, said Dr. Allen.

"Numerous efforts were made historically and none worked very well until the development of siliconized rubber tubes and very discreet types of valves — these beginning in the late '50s."

Siliconized rubber tubes are made of silicon tubing impregnated with silicon dioxide to make a long-lasting, non-irritating tubing, Dr. Allen said.

Shunts, however, are not without complications. The most common complication is infection.

"Anytime you put a foreign body into a human body you have the possibility of infection," he said. Other complications include blockage of the shunt, breakage of the tube and erosion of the shunt through the skin, as well as some scarring and seizures.

Robert's shunt malfunctioned and was surgically repaired when he was one. At 4, he again underwent surgery to lengthen the shunt.

Although Robert's hydrocephalus developed after birth, doctors can use ultrasound or other monitoring devices to spot a problem when it begins developing in the womb. "The first sign (in an infant), an enlarged head, can sometimes be detected before birth and we're often asked to be ready to do a shunt the day before (delivery)," Dr. Allen said.

The bones in an infant's head have not fused and expand easily, so the first visible sign is an enlarged head. This can be noted in the womb or at birth, or the head may look normal at birth and grow shortly thereafter.

"An infant, except to cry, has no way to complain," said Dr. Allen. But 'sunset eyes' (a downward turning of the eyeballs), stupor and vomiting are other signs that doctors look for.

Hydrocephalus in infants has several causes. About half of the infants treated for hydrocephalus in the MCG Pediatric Neurosurgery Clinic were born with myelomeningocele — a congenital problem that leads to hydrocephalus in nearly 75 percent of all cases. Other congenital problems such as blocked canals in the brain and abnormalities in the back of the brain can also be factors.

Non-congenital causes include blockage at any of several points in the system of channels that pass cerebrospinal fluid through and over the brain. The blockage may be caused by hemorrhaging or infection. Sometimes fluid may also be trapped between the brain and the membranous covering beneath the skull.

Hydrocephalus, however, isn't limited to infants, according to Dr. Allen. Older children and adults can develop the syndrome as a result of hemorrhages, tumors or brain displacement, in which the brain becomes dislodged and blocks the flow of fluid from the brain into the spinal column.

Hydrocephalus is usually much more chronic in adult cases, Dr. Allen said. Irreparable brain damage may occur because the adult skull bones have fused, leaving the head no room to expand. Pressure increases and the brain is more quickly compressed. The only outward signs of adult hydrocephalus may be headache, nausea, stupor and 'sunset eyes'.

The adult patient then suffers brain damage, loses coordination of movements and becomes incontinent, Dr. Allen said.

Adult patients are also treated with shunts to route the fluid from the head to another part of the body.

"The unfortunate thing is that the success rate for treating chronic hydrocephalus in adults is well less than half," Dr. Allen said. "The figures show a 20-40 percent rate of success."

Although shunting has been proven effective in moving fluid from the brain cavity into the bloodstream, problems in the final outcome relate to the amount of damage done before the shunt is administered. Retardation might remain. When diagnosed at an early stage, hydrocephalus can be treated more successfully, Dr. Allen said.

"We have two goals when treating children," Dr. Allen said. "Ideally, we'd like to have a normal lifestyle and certainly, the higher the intellect the better." The second objective is supporting nursing care for those with severe retardation.

Like most hydrocephalic patients, Robert will have a shunt for the rest of his life. But he says he really doesn't mind.

"I don't really feel it," Robert said. The shunt is also barely noticeable, appearing as no more than a large vein in the neck.

Having the shunt hasn't ever kept Robert from being active, his father said. Most patients have complete freedom of movement, Dr. Allen said. He does, however, advise against contact sports.

Most patients are followed routinely and, once stable, have check-ups every six months to a year.

Maintaining the shunt depends on problems encountered, and Dr. Allen said revisions in the shunt are necessary about every two or three years.

Over the years, doctors have developed better ways of routing the shunt. Robert's shunt begins in his head, travels along his neck and down to his abdomen — the most common route.

"We used to put tubes from the brain to the heart," Dr. Allen said. "We still do, but rarely, because there are more complications." In certain select cases, the shunt is placed to run from the spinal canal under the skin to the abdomen.

Thanks to improvements and changes in the shunts themselves, as well as the way they are administered, more and more hydrocephalic infants live normal lives like Robert, said Dr. Allen. Ninety-five percent survive 10 years or more, and 70 percent have normal intellect.

"Today, a much higher percentage have a normal life and intellect than in the past." ■

— Karin G. Calloway

Hope for Epileptics

A research scientist is transplanting healthy brain tissue into laboratory rats to see if it halts seizures.

Epileptic seizures are sneaky, vicious intrusions. They can strike at any time without warning and render victims debilitated.

Society often associates epilepsy with mental illness, and victims (1 to 2 percent of the population) pay the price socially and professionally. They can also pay with their lives: seizures can be fatal.

"Life is often a struggle for patients with epilepsy," said Dr. Gregory L. Holmes, associate professor of neurology and pediatrics at the Medical College of Georgia. "Even in this day and age, it's associated with many misconceptions, most of which are unfavorable."

Many children with epilepsy outgrow their seizures. But for those who don't, Dr. Holmes would like to eradicate their plight. And he's working to do just that.

Epilepsy, recurrent seizures which can be inherited or caused by some damage to the brain, is characterized by overexcited brain neuron activity. Dr. Holmes reasoned that replacing the damaged brain tissue with healthy tissue would eliminate the epilepsy.

To test the theory, he has administered a seizure-inducing drug to rats. He has just completed transplanting brain tissue of 17-day-old rat fetuses into the rats, and is waiting to see if the seizures will cease. If so, human epileptics may be next in line for brain tissue transplantation.

"It's something I think will be done in the future to repair tissue damage," he said. "Of course, a distinct possibility is that the transplants will be ineffective."

Even if the research is successful, Dr. Holmes acknowledged that moral and ethical implications loom large. Humans would have to receive brain tissue of human embryos, so donors would have to be the result of miscarriage or abortion.

But Dr. Holmes said he hopes people will understand the importance of the results and accept brain tissue transplants as they accept organ transplants.

Those who think brain tissue transplantation sounds like science fiction might be surprised to know that such research began around the turn of the century. Scientists transplanted tissue from one laboratory animal to another, but they didn't take the donor's age into consideration.

"They found that it just didn't work," Dr. Holmes said. "The brain tissue didn't grow."

They stumbled onto the solution in the 1920s when they used baby rats as donors instead of adults. The immature tissue survived and grew.

More recent research has yielded the knowledge that successfully transplanted immature brain tissue not only grows, but appears to alter brain abnormalities. The process also happens quickly.

Epileptics currently are treated with drugs, but Dr. Holmes said the success rate is only about 70 percent. The seizures that result from that 30 percent failure rate are costly, he said.

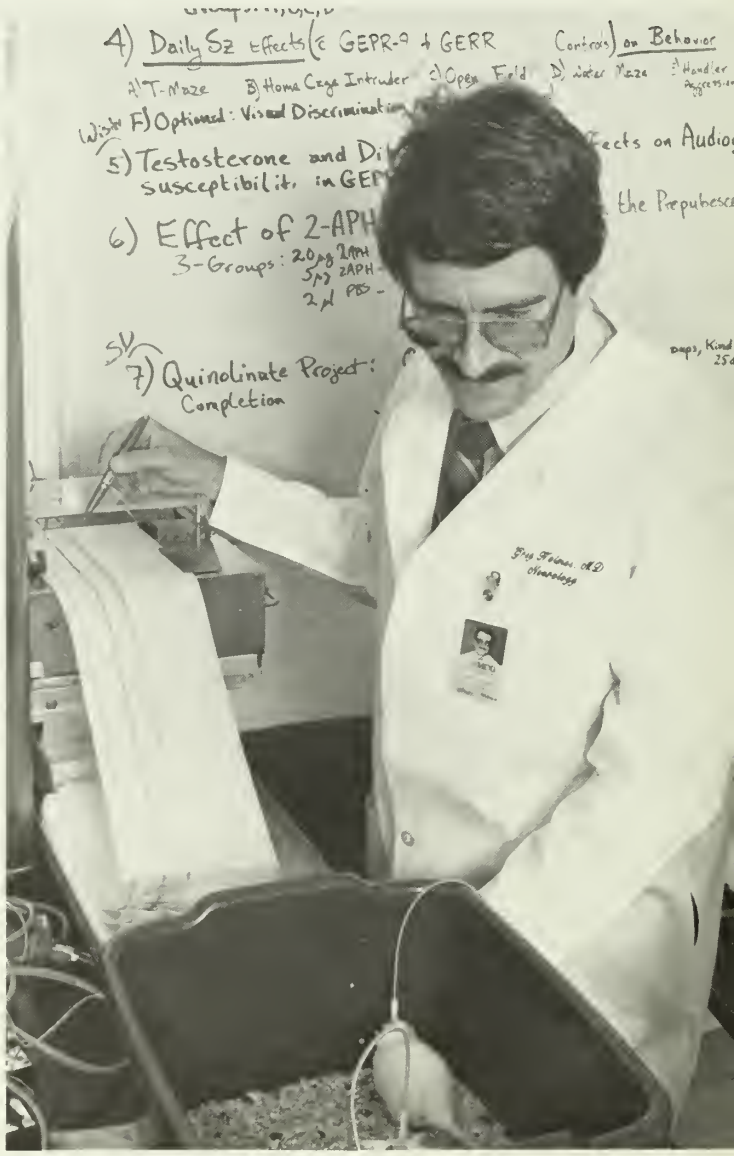
"Some people who have frequent seizures tend to go downhill and lose IQ points," he said, noting that it isn't known whether the lowered intelligence is a result of the seizures or the drugs.

Another alternative is surgery, in which the damaged portion of the brain is removed. But only a small percentage of epileptics are eligible since the damaged tissue must come from a well-localized area of the brain. Many epileptics have damage in areas that cannot be treated with surgery. Also, surgery can result in some memory loss.

Dr. Holmes is optimistic that tissue transplantation may help patients who can't be treated surgically. Even if his test rats' seizures continue — which he doesn't anticipate — he plans to continue the research.

"There are lots of variables we'd want to look at before we would give up," he said. "I think we have to stress that tissue transplantation for the treatment of epilepsy is very theoretical; we're years away — but I think it's a potential treatment." ■

— Christine Deriso



After transplanting healthy brain tissue from fetal rats into this adult rat, Dr. Greg Holmes tests the rat for evidence of seizures.

Mind Games

Neuropsychologists conduct brain tests to evaluate patients for epilepsy surgery.

"Keep your arm in the air," said neuropsychologist Dr. Gregory Lee.

A simple enough command, but more than Michael Angel could manage. The arm swayed erratically and dropped to his side.

"What is this?" asked Dr. Lee, holding up a picture of a pen. Mr. Angel struggled to respond, but couldn't.

The patient is epileptic, but these particular disabilities had nothing to do with the condition. They were drug-induced. The left side of Mr. Angel's brain was paralyzed with an injection of sodium amytal.

A few minutes later, after the medication had worn off, the right side of his brain was paralyzed, and the tests repeated.

"What is this?" repeated Dr. Lee, again showing him the picture of a pen.

"A pen," Mr. Angel responded groggily. Since he was able to name items and understand commands only when the left side of his brain was functioning normally, doctors determined that the left side controlled his speech.

In a matter of minutes, the medication had worn off, and Mr. Angel was back to his old self, no worse for the wear. The Wada test he had just undergone (named after the Japanese neuroscientist who developed it) was one of several used to determine the functional makeup of his brain. That information plays a crucial role in evaluating patients for epilepsy surgery.

Epilepsy, recurrent seizures caused by overactive neural activity in the brain, can in some cases be partially or fully remedied by removing the damaged portion of the brain.

The tests conducted by Drs. David W. Loring and Kimford J. Meador, assistant professors of neurology who are part of a large team of doctors evaluating epileptics at MCG, isolate the parts of the brain necessary for such vital functions as memory and language. Armed with this information, surgeons can remove the damaged portion and leave healthy, vital tissue intact.

The surgery isn't risk-free, but Drs. Meador and Loring agreed that it is a godsend for some epileptics.

"When people see seizures, they freak out essentially. There's such a social stigma," said Dr. Loring, a neuropsychologist. "I would say unequivocally, seeing the people (who have had surgery) and how well they do, I would go ahead and have it done myself."

Only about 5 percent of the brain (usually part of the anterior temporal lobe) is removed, and most patients suffer no complications.

This breakthrough probably wouldn't be an option without the preliminary tests performed by Drs. Loring and Meador.

"The tests provide very strong evidence that you're going to cut in the right place," Dr. Meador said.

Another test used to measure temporal lobe function is brain mapping, a procedure Dr. Meador researched which has now become routine when testing potential surgery patients.

In mapping, a computer analyzes brain wave responses to stimuli. The damaged part of an epileptic's brain doesn't respond normally, and the color-enhanced computer test indicates this with a darkened printout. A printout in shades of red, yellow and orange indicates healthy tissue. If seizures occur on both sides of the brain, which is unusual, both printouts are dark. If they occur in an area other than the anterior temporal lobe (also unusual), both printouts are colored.

The doctors also use scalp, sphenoidal and depth electrodes to study actual seizures. The electrodes are attached to the patient for weeks to determine accurate data about the seizures.

"The 'gold standard' for determining the part of the brain that's affected is recording seizure onset," Dr. Meador said.

Although the doctors usually feel confident about their patients' brain makeup after these procedures, tests continue during surgery. Different parts of the brain again are electrically stimulated, and memory and language tests can be repeated before the brain is cut.

How do patients feel about their brains being manipulated in these ways?

Before his Wada test, Mr. Angel was calmer than most people are before getting their teeth cleaned. Any hesitations or anxieties? None were evident. In fact, he was enthusiastically extolling MCG, calling it the best hospital he knew of, one he bragged about to his friends. He said he was glad to undergo the tests. The brain surgery for which they were preparing him would likely help or cure his epilepsy, and that was the important thing, he said.

"Most of these people have had seizures for so long, they're more than willing (to undergo the tests)," Dr. Loring said. "They're willing to try anything." ■

— Christine Deriso

Dr. Kimford Meador conducts a Wada test to reveal the functional makeup of patient Michael Angel's brain.

Happy Birthday

The Medical College of Georgia plans to celebrate 160 years of teaching, discovering and caring during 1988.

In 1829, the Medical Academy of Georgia was seven students attending lectures in the back rooms of a city hospital on Greene Street. They studied chemistry, midwifery and diseases of women and children, anatomy and surgery. Their teachers were Milton Antony, Ignatius Garvin and Lewis Ford, great names in the history of the Medical College of Georgia.

Only the year before, Dr. Antony had persuaded the Georgia General Assembly to charter a medical academy with the authority to offer a one-year course leading to the bachelor of medicine degree.

Today, MCG is a 93-acre campus with 640 full-time faculty and about 2,300 students studying for degrees not only in medicine but also in nursing, allied health sciences, dentistry and graduate studies.

As the state's health sciences university, MCG looks back with pride on 160 years of teaching, discovering and caring. During its 160th anniversary year, those three missions will be in the spotlight.

The official kick-off of MCG's year-long 160th anniversary celebration was at a January reception attended by members of the Board of Regents and state and local legislators, among others.

Each of the five schools and the Student Government Association will have a special committee to plan monthly activities related to the anniversary celebration.

People around the state will also have the chance to get to know MCG through special radio and television spotlights on great moments in MCG history and through live interviews of MCG scientists and others. Chambers of commerce in Georgia will have access to a 10-minute videotape on the past, present and future of the Medical College of Georgia.

A collection of MCG memorabilia will also be offered to employees, alumni and visitors throughout the year: glassware, visors, ink pens, T-shirts and more. Alumni will also be able to join the celebration through regional alumni dinners and activities during Homecoming '88.

The 160th anniversary will mean more than a year of celebration in the long run. A five-year image enhancement plan for MCG begins during 1989. Details will be announced later. ■

Students in the dissecting room ca. 1896-7 (right). Class of 1951 poses on the steps of the Old Medical College building (below).

Preserving the Medical College of Georgia's Heritage

A fund-raising campaign during 1988 will support the renovation of the Old Medical College building.

A fund-raising campaign to finance the renovation of the Old Medical College building has reached more than $800,000 of its $1.2 million goal, according to John Anderson, vice president for development at the Medical College of Georgia.

The campaign is continuing, Mr. Anderson said. Major activities will include a phone-a-thon, mailings and personal solicitations.

The renovation, which will span approximately one year, is being directed by Atlanta architect Norman Askins, who has extensive experience in historic preservation. He has worked on the preservation, restoration and adaptive use of historic buildings in the Southeast since 1977 and also worked on a year-long project involving the redesign of the executive office wing of the White House.

A committee made up of members of the Medical College of Georgia Foundation Board of Directors John C. Hagler III, Dr. William B. Mullins and Whitney C. O'Keefe and Mr. Anderson, executive director of the foundation, will oversee the renovation and operation.

Mr. Askins, structural engineer Tom Robertson and contractor Lawrence Smith have developed renovation plans in four areas:

☐ Rewiring, replumbing, sprinklers, air conditioning and heating, installation of audio-visual systems.

☐ New wood floors, wall repairs, new light fixtures, new stairs, elevator, reopening of rotunda, renovation of kitchens.

☐ New roof and guttering, new stucco and exterior waterproofing, paving and landscaping of parking area.

☐ Furniture, draperies, medical history displays, paintings and other interior design work, landscaping and outside lighting.

The building will be open for the President's Reception during Homecoming weekend May 6-8. When renovations are completed in late 1988, the building will be used as a center for continuing education, alumni and community activities.

"The building is structurally in absolutely astounding condition," said Mr. Askins.

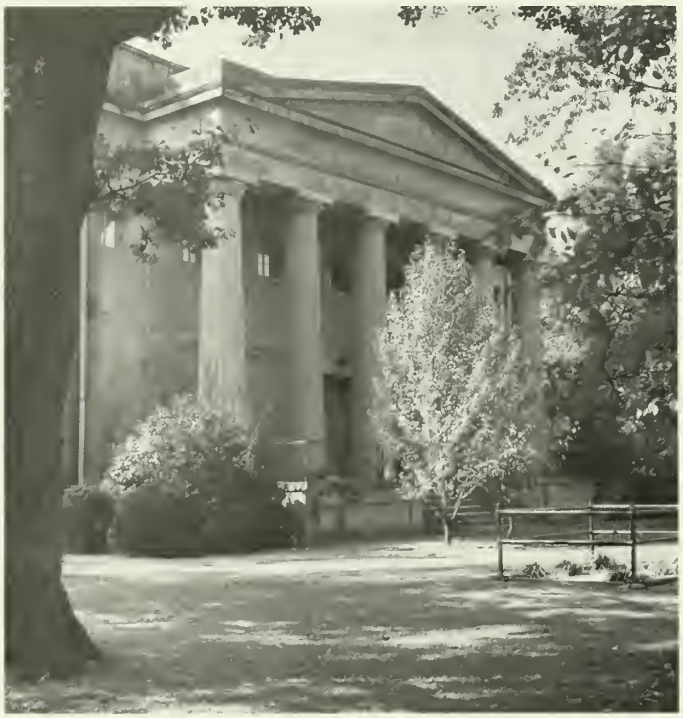
Norman Askins with an architectural rendering of the Old Medical College circa 1934.

The Old Medical College is not only one of Augusta's finest buildings, but is also "a nationally important building," he said.

Built in 1835, the Greek Revival building is one of America's earliest structures designed by a professional architect, Charles B. Cluskey. Mr. Cluskey later designed renovations for the U.S. Capitol and the White House.

As an example of early American neoclassical architecture, the Old Medical College is unsurpassed for purity of design, Mr. Askins said. Its clean lines, absence of ornamentation and emphasis on form typify the style of an early movement toward establishing a national architecture, an endeavor undertaken by Thomas Jefferson and early American architect Benjamin Latrobe.

The Old Medical College was a teaching facility until 1912, when the medical school moved to a former orphanage remodeled to accommodate the growing college. The building, constructed on land leased from Richmond Academy, then reverted to the academy. The academy held classes there between 1914 and 1926, and the building was then vacant for five years. In



Old Medical College building

1931, the Sand Hills Garden Club rescued the building from virtual demolition by vandals, and it was used by various civic and social organizations during the '30s. The building housed a U.S.O. canteen during World War II and has been maintained since 1948 by the Augusta Council of Garden Clubs, which has used it as an activity center.

Donors who contribute toward the renovation will be recognized according to the amount of the gift. Those who give $1,000 or more will receive a leather bound, signed edition of *The History of the Medical College of Georgia* by Phinizy Spalding. Those who give $2,500 or more will be recognized by having their names engraved in bronze on the master plaque in the foyer of the building. Major contributors may reserve a named room or area of the building. These include:

☐ Foyer, $150,000 — A prominent and spacious entrance area.

☐ Rotunda, $70,000 — The focal point of the building, accessible through a balustrade on the second level.

☐ Lecture Hall, $100,000 — May be used as a classroom or auditorium. Chairs can be removed for receptions. Space for up to 150 people.

☐ Seminar Room, $75,000 — For meetings, seminars and small receptions. Space for up to 50 people.

☐ Conference Room, $50,000 — A meeting room with elegant furniture and paintings. It adjoins the seminar room.

The solarium, ballroom, library and courtyard have been reserved.

More information about the renovation and fund-raising campaign is available in a brochure which can be obtained by calling the Medical College of Georgia Foundation at (404) 721-4001. ■

Gifts of Securities

Donating highly appreciated securities has always been among the most economical ways of making a charitable contribution. The Tax Reform Act of 1986 has only made a good situation better.

Tax reform eliminated the favorable capital gains treatment that applied when selling appreciated securities which had been held for more than six months. That former rule made 40 percent of the gain subject to the tax at a maximum rate of 50 percent. The highest possible tax was then 20 percent of the gain.

Effective last January 1, all gains realized from the sale of securities are treated as ordinary income and are subject to one's normal tax rate. The highest 1987 tax rate is 38.5 percent, meaning that for some taxpayers the applicable rate has almost doubled.

A gift of securities held for more than six months in your annual contribution budget may be advantageous. First, the transaction qualifies for the charitable deduction which is the market value on the date of the gift. This means a deduction of your original cost plus appreciation.

A second consideration is that such a gift may avoid the entire tax on that appreciation. Thirdly, transferring a gift of securities is almost as easy as writing a check.

If you want to continue to hold a position in a particular stock for further appreciation, donate that stock and purchase replacement shares which would increase their basis to current market value. This will reduce your future taxes when you sell them.

Another possibility exists with highly appreciated securities which pay a low dividend yield. You face a choice between selling and paying the tax on the gain or continuing to receive the low dividends. A gift of those securities to the Medical College of Georgia Foundation in exchange for a guaranteed life income contract will accomplish these goals: (1) Avoids income tax on the gain; (2) Provides a fixed income — probably higher than the dividends; (3) Allows you to provide substantial support to a program at the Medical College of Georgia with the remainder of the funds at your death; and (4) Eliminates your concerns over future management of funds.

One aspect of the 1986 Tax Reform Act is that appreciation on the contributed securities is considered a preference item to be included in the Alternative Minimum Tax test. For the most part, donors who do not have other tax preference items will not be subject to the Alternative Minimum Tax. Your tax adviser will be able to offer suggestions on that matter.

If you have any further questions regarding planning your charitable gifts, please call Bruce L. Howerton, director of planned gifts, at 404 721-4002 or write Mr. Howerton at the following address: Alumni House EA-100, Augusta, GA 30912-5000. ■

Omission noted

The Augusta Junior Woman's Club was inadvertently omitted from the donor recognition section of our 1987 Annual Report issue. Through their generous contributions, they attained membership in the President's Club — Partner Level. We are sincerely grateful for their support and regret this error. ■

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